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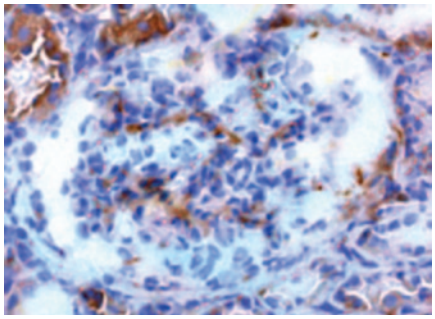
Abnormal glycosylation of IgA in cirrhosis



Deposition of IgA in the glomeruli in some patients with alcoholic cirrhosis has been well recognized. It is now clear that, in IgA nephropathy, the IgA molecules lack the terminal galactose and sialic acid, which are added to *N*-acetylgalactosamine. These sugar abnormalities likely play a very significant role in the formation of deposits on the mesangial cells. In IgA nephropathy, this abnormally glycosylated IgA also causes proliferation of mesangial cells *in vitro*, suggesting that it might play a role in pathogenesis. Tissandié *et al.* show that patients with advanced alcoholic cirrhosis have the same anomaly in their IgA molecules. The IgA is shown to bind to human mesangial cells but does not seem to induce proliferation. The abnormally glycosylated IgA in IgA nephropathy has recently been shown to be inherited in IgA nephropathy. The paper by Tissandié *et al.* suggests that environmental influences might also cause this, but more work needs to be done to rule out inherited susceptibilities in the patients with cirrhosis who develop IgA glycosylation anomalies. **See page 1352.**

Sickle-cell trait and progression of renal disease

Sickle-cell disease is associated with chronic kidney disease (CKD) and progression to renal failure, and the association is likely to be causative. Homozygous mutations result in sickling, especially in the medulla, with consequent occlusion of the vasa recta and tubulointerstitial damage. Hicks *et al.* addressed the question of whether the heterozygotes with sickle-cell trait are also associated with renal disease or progression of renal disease caused by another condition. They used a large database of African Americans and found that the trait was present in about 7–8% of the population. They also tested for the presence of variants of *APOL1* and *MYH9* that are known to be associated with renal disease. There were close to 1000 patients with type 2 diabetes in the cohort studied. While it is obvious that these conditions are all associated with a risk of end-stage renal disease (ESRD), there did not appear to be an influence of the presence of sickle-cell trait on the presence of CKD or ESRD in any of these conditions. Hence, the message of this article to the large population of subjects with the trait is that the presence of this heterozygous mutation does not add to their risk of renal disease. **See page 1339.**

Objective and perceived knowledge of CKD by patients

Communications between patients and physicians are in a sorry state, especially in the case of chronic diseases such as CKD. As they report in this issue, Wright Nunes *et al.* quantified this dismal state using formal testing of more than 300 patients with CKD. Shockingly, almost three-quarters of the patients did not have a clear understanding of their medications and how or whether they could help their kidney function. An interesting aspect of this study is the comparison between objective knowledge, what patients actually know, and their perceived knowledge, what they think they know. This study, full of the statistical analysis that sometimes can hide the basic piece of information, has a simple message: patients need education regarding their disease, as this will increase their involvement in the way the disease is managed. An added benefit is the likely increase in patients' satisfaction with the care delivered by their nephrologists when their objective knowledge increases. **See page 1344.**